

Introduction to toxicology



- Toxicology :- the science of poisons, the study of the Adverse effects of chemicals or physical agents on living organisms

★ all things are poison, solely the dose determines.

- classification of Toxic Agents

- ↳ physical state [gas, dust, liquid]
- ↳ chemical stability [explosive, flammable, oxidizer]
- ↳ chemical structure [aromatic, halogenated, C≡O]
- ↳ poisoning potential [extremely, very, slightly]
- ↳ Biochemical potential [alkalizing, cholinesterase]

★ Toxic response are related to Dose [دوز] except for

- ↳ Allergy → we all know حساسية
- ↳ Idiosyncratic reactions
 - ↳ weird, unpredictable genetically based drug reaction

- chemical Interactions

- ① Additive :- mc type [2+3=5]
- ② Synergistic :- [2+3=10]
- ③ potentiation :- [0+3=10]
- ④ Antagonist

Type	Meaning	Example
Receptor antagonism	Two drugs act on the same receptor — one activates it (agonist) and the other blocks it (antagonist).	Adrenaline (agonist) vs. beta blockers (antagonist).
Functional antagonism	The two drugs act on different organs or receptors but produce opposite physiological effects .	Adrenaline increases blood pressure, while histamine decreases it.
Chemical antagonism	The drugs chemically react with each other and form an inactive compound.	Antidotes that neutralize poisons, e.g. chelating agents for arsenic.
Dispositional antagonism	One drug alters the absorption, metabolism, or excretion of another, reducing its effect.	Activated charcoal reduces absorption of poisons from the gut.

Principles of Management of Acute poisoning

- Sources [House products, Food poisoning, Environmental, Occupational, Substance abuse]
- Routes [Oral, Inhalation, Parenteral, Dermal]
- Circumstances [Accidental, Suicidal, Homocidal]
- General approach to management **6 points**

- 1 Patient stabilization :- ABCD (+) Antidote
- 2 Complete patient assessment :- Hx + PE + Labs
- 3 Poison decontamination

Methods of GIT decontamination

- ↳ Dilution :- giving water or milk to reduce the poison
- ↳ Emesis :- Inducing vomiting
 - Ipecac syrup :- orally, on vomiting, safe center
 - Apomorphine :- given by injection, rarely used, cause CVS depression
- ↳ Gastric lavage :- using 1L of saline in hospitals, have CX
 - Soft liquid detergents :- Irritation of the stomach lining, 15-30 ml, safe
 - Gurg reflex :- mechanical stimulation, cause trauma
 - Hypertonic solution :- High sodium or Bicarbonate or salt intake, cause electrolytes imbalance
- ↳ Adsorbents :- substance that binds the poison in the stomach, ex -> Charcoale
- ↳ Cathartics :- Increase bowel movements to speed up the elimination, cause organs failure
 - Dose 1-2 gm/kg, Adults: 50 - 100g
 - Child: 15 - 20g

Poison enhancement of elimination

- ↳ Urin
 - Alkalinisation by sodium bicarbonate
 - Acidification by Ammonium chloride
- ↳ Cautions to pulmonary edema, cerebral edema, Electrolyte imbalances
- ↳ Dialysis ->
 - small M.W
 - water soluble
 - low protein binding
- ↳ Hemodialysis better than peritoneal dialysis
- ↳ Indicated in renal failure ppl

5 use of poison antidote

6 Continuous patient supportive care



- * Ethanol $\text{CH}_3\text{CH}_2\text{OH}$
solvent and fuel found in Distilled spirits, Wines, Beers, Mouthwashes, colognes and medicinal preparations
- * Pathophysiology of Ethanol
 - ↳ Acute effect \circ - CNS depressant, frontal then occipital lobes
 - ↳ chronic effect \circ - \uparrow uric acid

→ hallucinations
هلوسات بصرية
- * Absorption of Ethanol
 - ↳ rapidly absorbed in oral mucosa \rightarrow gastric mucosa \rightarrow small intestines
 - reaches a peak after 30-60 minutes
- * Distribution of Ethanol
reach BBB and placenta, in women less
- * Elimination
 - 10% by the liver 90%
 - 20% by the kidneys 10%
- * Manifestations التزيب المزب
 - ↳ \downarrow Glucose in children
 - ↳ CNS depression, irritability, drowsiness, stupor and coma
 - ↳ RS depression and hypoxia
 - ↳ change in mental status
 - ↳ Hypothermia
 - ↳ Cognitive and motor skill abnormalities
 - ↳ slurred speech
 - ↳ trauma
 - ↳ dysrhythmias
 - ↳ strokes, malnutrition, obesity, vitamin \downarrow
 - ↳ Urticaria
 - ↳ inhibition of spermatogenesis
 - ↳ fetal alcohol syndrome

* Neurologic impairment depends on → genes, الكمية, prior, other drugs
experience

* Disulfiram reaction: - When taking drugs with alcohol

ازول ← azole و مدرات المعدة و فلاجيل

Sulfonamides hypoglycemia agents

بغير عند الشخص استفراغ رعبية رتارغ



* Laboratory investigations

↳ glucose يكون نازل

↳ Electrolytes levels

↳ Ethanol levels by urine or breath

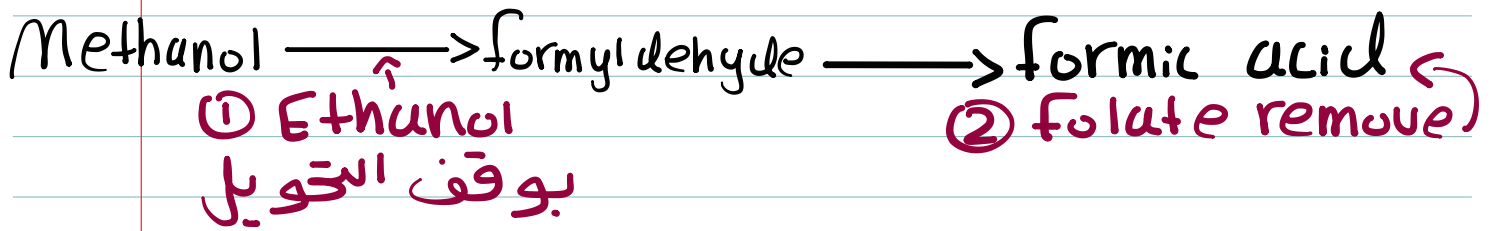
* Treatment اوجيني جدا سكران وبدل تصحيحه شو يتعمل؟

Correct hypoglycemia, and correct electrolyte, give Thiamine

Methanol

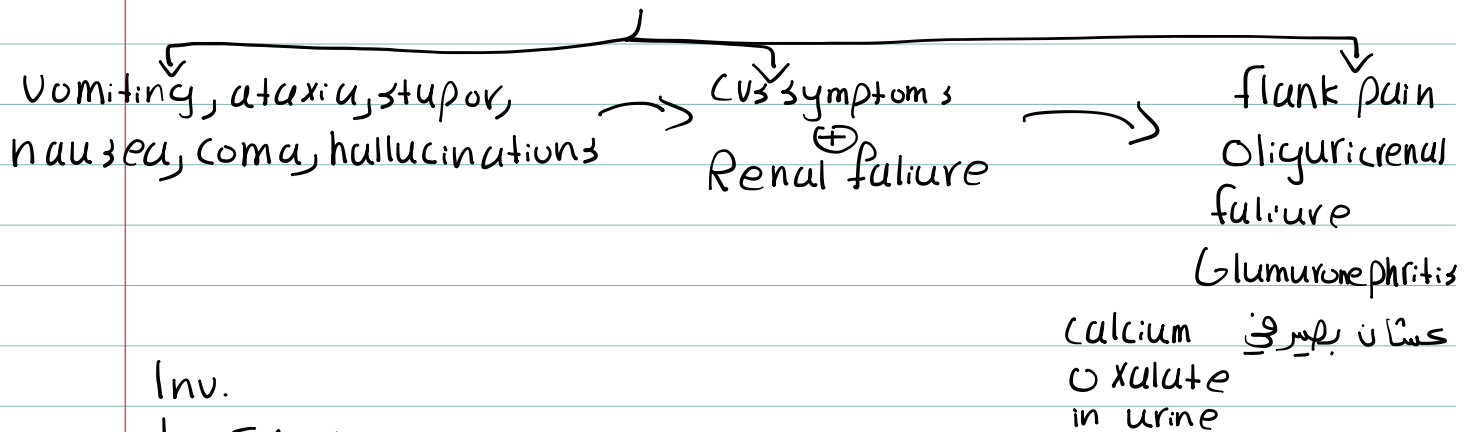
- colorless, Gas, Industrial uses
- CNS depressant similar to ethanol
- Absorbed by GI, skin and lungs
- Distributed by GI, liver, kidneys
- Eliminated mostly Hepatic
- clinical presentation
 - ↳ CNS depression
 - ↳ Cerebral edema
 - ↳ Eyes: blurred vision, photophobia, retinal edema
 - ↳ metabolic acidosis, dyspnea, shock, coma, seizures
 - ↳ Death from respiratory arrest
- management كذا افقرض الميثانول
 - ↳ Gastric lavage, Hemodialysis

قبل فاتفهم Anticlot's المستخرجة افهم pathway 3



Ethylene Glycol

- odorless, colorless, nonvolatile liquid, having a sweet taste
- Weakly toxic, but cases of poisoning are common
حارة "خزنوه بكراجات البارات فالهغار كانوا بيثوفوه لونه اظفر ولعمه نوعا فاشناز
فكانوا بيتمهوا فيه
- Clinical Manifestation [3 phases]



Inv.

- > EG levels
- > ABG
- > ECG
- > Urine analysis
- > kFT

calcium oxalate in urine
حسان بهير في

Treatment

- > correct acidosis
- > Monitor cardiac function
- > Ethanol or fomepizol
- > Gastric lavage
- > Hemodialysis
- > pyr. doxine and thiamine

4

Doping in sports ★

* What is doping?

the use of prohibited substances or methods to enhance an athlete's performance.

• Classification of substances in Doping

- ↳ Always prohibited
- ↳ prohibited during activity
- ↳ prohibited in specific activity

• Why do they take them?

- ↳ to improve performance
- ↳ Increase alertness
- ↳ Improve appearance

• Methods of Doping

- ↳ pharmacological
 - ↳ hormonal
 - ↳ Non hormonal
- ↳ Non-pharmacological
 - ↳ Blood transfusion
 - ↳ Hypoxia induction
 - ↳ Gene doping

• performance enhancing drugs

I Anabolic steroids

* male hormone testosterone

growth of muscles, bones, ↓ fat

↑ metabolic effect ↑ فاعلية ↑

↳ Androgenic effect
 ↳ صفات الذكورة باختصار

and have also medical uses

- Administered by many routes
- use type [Cycling, stacking, pyramiding]
- Adverse effect: MC is hypertension

Testing

Normal 4:1
 Positive 6:1

• After AAS, over 50% used drugs such as:

- 31% estrogen receptor inhibitors to prevent breast enlargement
- 22% HCG ⊕ testosterone production
- 17% diuretics and/or "uppers" to lose water
- 15% pain killers to manage pain after exercise

② Human Growth Hormone

- Increases body weight and muscle mass
- give by IM injections
- very expensive!
- side effects:- سكري, تآخيم, Cardiomegaly, Hypogonadism, myopathy

③ Narcotics

- used to reduce fever or swelling
- Dependence

④ Stimulants

- Amphetamines [Most potent ergogenic drugs in the stimulant category]

Adverse effects : Heat stroke, Addiction, Withdrawal syndrome, Depression, ↓ athletic performance

⑤ Beta Blockers [propranolol]

- it decrease hr, hand tremor, ↓ anxiety so it gives you a steady hand
- prohibited
- Adverse effects : Bronchospasms, CVS disturbances, hypotension, impotence.

⑥ Diuretics

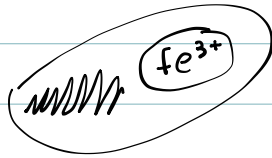
cause dehydration, ↓ Blood volume, Muscle cramps, Renal disorders, Dizziness, Disrupted Heart rhythm

⑦ Erythropoietin and Blood Doping

- to improve the aerobic capacity and muscle endurance
- It cause bacterial infections, shock, HTN, stroke, ↑ viscosity of the blood

6 Cyanide poisoning

- Sources \Rightarrow seeds of apple, peach, plum, apricot, cherry
- Cyanide \Rightarrow hydrocyanic acid, prussic acid
- Mechanism of toxicity:-
Histotoxic anoxia but how?



this is a mitochondria that have an enzyme called cytochrome oxidase, who have Fe^{+3} الإنزيم
When cyanide binds the cell is inhibited and there is no use of the oxygen \longrightarrow so cellular hypoxia & anaerobic metabolism & metabolic acidosis

Cyanide detoxification & excretion

Pathway	Product	Excretion Route	Importance
Rhodanese + thiosulfate	Thiocyanate (SCN^-)	Urine	Major
Cobalamin (Vit B ₁₂)	Cyanocobalamin	Urine	Minor
Direct excretion	CN gas	Breath & Sweat	Minor

- Signs and symptoms of cyanide poisoning
Weakness, Dizziness, Headache, N&V, Tachycardia, flushing

Treatment

ABC

Decontamination

Antidote [Amyl nitrate, sodium nitrate, sodium thiosulfate]

Continuous care

★ Substance Abuse / CNS Depressants

- Substance Abuse:- the use of a substance for its pharmacological effects without medical indication
- Problems of method of abuse:-
 - ↳ Injections:- infections, aids hepatitis
 - ↳ Snorting:- suffocation
 - ↳ Smoking:- pulmonary problems, cancer
 - ↳ social and financial:- loss of money, crimes

- What are Depressants
 - ↳ Narcotics & Drugs to relieve pain
 - ↳ Depressants & مخدرات

1) Narcotics

- ★ - they are drugs used to relieve pain, have high potential for abuse
- ★ - Possible effects of using them
 - ↳ Euphoria, drowsiness, respiratory depression
 - ↳ constricted pupils
 - ↳ ADH, constipation
- ★ - Method of abuse
 - ↳ usually injection
 - ↳ smoking / heroin
 - ↳ oral / methadone
- ★ • Management
 - ↳ supportive
 - ↳ Antidote
 - ↳ treat withdrawal

2) CNS Depressants

- 1) Opioid
 - ↳ Naturally
 - ↳ semi-synthetic
 - ↳ synthetic
 - ↳ Antagonists

- ★ Lets talk more about Heroin
 - HL → 30 min
 - duration of action → 4-5 hrs
 - lipid soluble that reach BBB
 - clinical presentation
 - ↳ blurred speech, appear sedated, pinpoint pupils
 - PE :- marks التهاب, subacute bacterial endocarditis, lymphadenopathy, huge liver

* Management / opioid Intoxication

1) opioid agonist tx => Methadone, Buprenorphine

2) Antidote => Naloxone

3) psychosocial support

* Withdrawal syndrome

↳ high dependence

↳ Watery eyes, runny nose, yawning, cramps

↳ loss of appetite, irritability, nausea

↳ Tremors, panic reaction, chills, sweating

↳ Convulsion, death

Stimulants - substances that induce a number of characteristic CNS effects include alertness with increased vigilance, a sense of well being, and euphoria

Examples => Amphetamines, Cocaine, Caffeine, Nicotine

Clinical presentation => Elevated mood, increased alertness, increased energy, insomnia, anorexia.

Physical Exam => HTN, ↑HR, pupillary dilation, hyperthermia, ↓Na, arrhythmias, MI, hemorrhagic stroke.

Mental Status

- Attitude - Tense, anxious, restless, agitated
- Psychomotor activity - Increased; dyskinesia
- Mood/affect - Good/euphoric or irritable and labile
- Speech - Talkative
- Thought processes - Flight of ideas; tangentially
- Thought content - Paranoia; auditory, visual, and tactile hallucinations; grandiosity; homicidal ideation
- Insight or judgment - Impaired
- Orientation - Confusion, delirium
- Memory - small doses may improve alertness and task performance, the heavy use associated with stimulant abuse can be detrimental to memory

Withdrawal

- Behavior - Sedated
- Psychomotor activity - Decreased
- Mood or affect - Depressed or irritable
- Speech - Decreased production
- Thought processes or content - Linear, at times with suicidal ideation and drug craving. Homicidal ideation; paranoia
- Insight or judgment - Variable
- Orientation - May be normal or close to normal
- Memory - Likely impaired due to sleep deprivation, associated fatigue, decreased attention and irritability

Method of Abuse

↳ swallowed, snorted, injected, or smoked

new type

Cocaine

• atropane ester alkaloid found in leaves of the Erythroxylum Coca plant, a bush that grows in South America

- Cocaine forms → cocaine base: smoked
↳ cocaine salt: injected or insufflated

• MOA of Cocaine: - increase dopamine اهرمني، تعرفوه

↳ blocking sodium ion channels so it cause cardiac arrhythmias
↳ local anesthetic effect

صا بجرق حفظ اولاً (٦)

Routes of Admnisitation

Route	Onset	Peak Effect (min)	Duration (min)	Half-Life (min)
Inhalation	7 s	1-5	20	40-60
Intravenous	15 s	3-5	20-30	40-60
Nasal	3 min	15	45-90	60-90
Oral	10 min	60	60	60-90

- Distribution : very Rapid to all organs

- Metabolism : 45% In the liver

الله يعين liver متويدة ليحل ليتكامل

- Elimination : In the urine

• Cocaine Intoxication

↳ CVS :- chest pain, arrhythmias, thrombus

↳ CNS :- agitation, seizures, coma, headache, ephorius سبب الغمالي

↳ RS :- Angioedema, pneumothorax, SOB, hemoptysis, wheezing

↳ GI :- ↓ salivary, ulceration, infarction, perforation, ischemic colitis

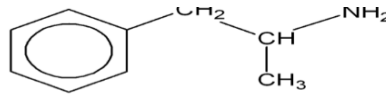
↳ skin :- pseudovasculitis lesions

Cocaine Management

↳ Supportive ABC → systems like cardio → No Antidote found.

new
typ

Amphetamines



• Target organs:

- CNS → stimulation 'euphoria, agitation, convulsion, tremor
- CVS → tachycardia, HTN, arrhythmia, collapse
- Other systems to be affected: endocrine, GI, skin, genitourinary
- Pregnancy → Spontaneous Abortion, Teratogenic

• Metamphetamine [crystal Meth] نوع قوي
used to treat ADHD wow!

Acute Clinical Presentation

- Increased energy and alertness
- Decreased need for sleep
- Euphoria
- Sweating
- Disrupted sleep patterns
- Tightened jaw muscles
- Grinding teeth
- Loss of appetite, contributing to weight loss with chronic use
- Disorganized thinking
- Itching
- GI symptoms such as nausea, vomiting, or diarrhea
- Dry mouth leading to serious tooth decay with chronic use
- Changes in mood consisting of irritability, anxiety, aggression, or panic

Management

- **General approach:** Control of agitation and other signs of sympathetic excess
 - Supportive care: ABC of Medicine
 - Care for cardiac complications, arrhythmia, hypertension, hyperthermia, convulsion, agitation, irritability, nutritional status
 - No definitive role for antidotes
- Assess the hydration and nutritional status.



Hallucinogens

Cannabis القنب الهندي

- have 2 forms → Marijuana
→ Hashish
- Absorption: - very fast and last for 2-3 hours
- taken by Inhalation
- High lipid soluble
- pathophysiology -



In short, THC from cannabis activates brain CB1 receptors that changes neurotransmitter release that causes mood perception and coordination effects.

• Manifestations [according to the dose]

Low 2mg	Moderate 5-7	High >15
Relaxation	thoughts فترتبتين	paranoia
Euphoria	Ataxia	Tachycardia
↑ perception لكواس كرفهم	short term memory impairment	↓ libido بدون راعها

- But guess what? ادقان نفسي وجبري
- heavy users if they stop it, they will have physical and emotional symptoms.

Acute poisoning

**Very rare (after Intravenous injection)

- N & V & D
- Abdominal pain
- Fever
- Hypotension
- Pulmonary edema
- Acute renal failure
- DIC
- Death

Investigations

- **Urine testing**
- Cannabinoids can be detected in the urine for as many as 21 days after use in persons chronically using marijuana,
- **Blood testing:** measuring the quantitative level of THC can distinguish between recent use and residual excretion

* Pesticides المبيدات الحشرية

• they are chemicals have been used to kill or control unwanted pests.

We also have Insecticides and Rodenticides



insects ←

→ rats, mice, moles



and also Herbicides, fungicides, fumigants
(:) medicides هراصة لا زونا

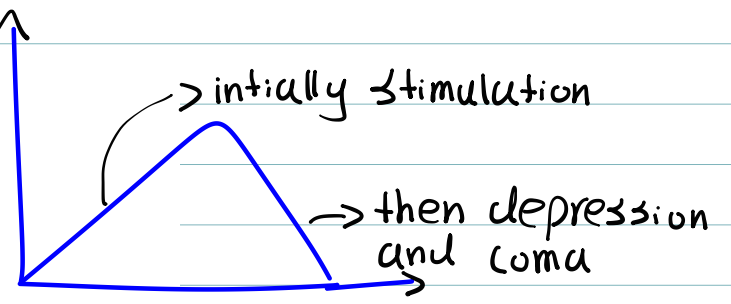
[Insectides]

pathophysiology: phosphorylation and deactivation of AChetylcholine enzymes.

clinical effects → M, constriction of pupils and contractions
→ N, persistent depolarization of skeletal muscles.
→ CNS,

Central Nervous System

- Anxiety
- Restlessness
- Tension
- Headache
- Ataxia
- Generalized weakness
- Convulsions
- Depression of respiratory => cyanosis



How to know this person؟ وكنتمهم

- ① odor => garlic like odor
- ② skin => dermal irritation
- ③ lungs => edema

اعراض فورانية يسبب
الاستخراجه لا ولافرة

↳ but on chronic use: - polyneuropathy, Neurobehavioural

• Labs \Rightarrow Red Blood Cell cholinestrase

بكون نازل

• How to asses \Rightarrow وريهن وشمم؟

① Stabilization \Rightarrow Intubation, give O_2

② Decontamination

③ give Atropine

المبيدات تبعمل overstimulation عشان هيلك بغيري
block عشانه بيجل for the receptors
للمستقبلات

for diagnosis \rightarrow IV. 1mg

for therapy \rightarrow IV. 2-4mg

④ give pralidoxime along with atropine

[μ receptors \leftarrow Atropine \leftarrow هيب عشانه فانخريل
N receptors \leftarrow pralidoxime]

⑤ Supportive care

Carbamates: اهدر للتسمم اذو, but less sever and shorter period.

• Organochlorines اهدر تسمم جريد

• Example \rightarrow Lindane is a garden spray

• CNS stimulators, affect CVS, and cause seizures

Management: - Diazepam, IV lines, CVS monitoring, skin decontamination

Acetaminophen Toxicity

Why we use paracetamol?

- Analgesic وسكن آلام
- Antipyresis خافض حرارة
- anti-inflammatory مضاد التهاب

* Dosing

↳ Therapeutic → ped: 15 mg/kg, every 4 hours, 5 مرات
 ↳ Adults: 1 gram every 4 hours, 4 مرات
 ↳ Toxic → Acute
 ↳ chronic

- ped can tolerate paracetamol more than adults
- febrile ped at higher risk of getting the toxicity

• What we mean by paracetamol toxicity?

We mean hepatic toxicity like: hepatic failure, encephalopathy, and death within days.

تسمم الإيسيتامول بتبسات حيت 😊

Pathophysiology of Acetaminophen (Paracetamol) Toxicity

- Normally, acetaminophen is metabolized by conjugation (glucuronidation & sulfation).
- In overdose, these normal pathways become saturated.
- More of the drug is converted to a toxic metabolite called NAPQI.
- Glutathione, which detoxifies NAPQI, becomes depleted (<30%).
- Excess NAPQI then binds to cellular proteins, damaging liver cells.
- This leads to cell injury and death → hepatic necrosis (liver failure).

👉 In short:

Overdose → more NAPQI → less glutathione → liver cell damage and death.

phases of toxicity			phase IV
phase I 0-24 hours silent overdose N&V & Abd pain	phase II 24-72 hours liver injury ↑LFT, PT, bilirubin	phase III 3-4 days N&V & Abd pain AST/ALT 10,000 encephalopathy coma	>4 days Recovery LFTs ↓

- It also could cause Renal toxicity
Try to P450 activity in the kidney
- The Normogram
It is a chart used to decide who needs treatment after a single acute ingestion, it does not show how fast the drug is eliminated from the body, only one level is needed, should be taken 4 hours after ingestion.
- In the U.S., the treatment line is set 25% lower to make it more sensitive, so no cases are missed

حالات رخصي لازم تتعا بع ٥-

Ingestion of single dose

Treatment indicated if:

- Level above 150mg/dL at 4 hours
- Ingestion of 150 mg/kg in children
- Ingestion of 7.5 g in adults
- Patient is unreliable or unconscious

So what we give in Paracetamol toxicity is

N-acetylcysteine

- time within 8 hours, but even if given late it helps.

Oral N-acetylcysteine

- Oral loading dose is 140 mg/kg
- Dilute 4:1 with palatable liquid
- Repeat doses are 70mg/kg every 4 hours
- Total of 17 doses for total of 72 hours
- Antiemetic treatment may be required
- NAC is very foul "rotten egg" liquid

IV N-acetylcysteine

- Can cause anaphylactoid reaction
- Rash, hypotension, bronchospasm and death
- Rate related; rare when given slowly
- Higher, continuous blood levels obtained than oral NAC
- Bolus administered first, then constant infusion rate may be given

IV vs. Oral

- Both have their advantages and disadvantages
- Each may be more appropriate in certain settings
- No side by side studies to date
- Conclusions of relative benefits are speculative

Take-Home Points

- "Rule of 150's"
- >150mg/kg = toxic dose
- 7.5g in adults
- >150mg/dL at 4 hours
- NAPQI and NAC: what they do
- Nomogram for single acute ingestions
- Very conservative but safe
- Treatment: indications, timing

How to manage?

①

Alcohol

- ↳ Ethanol → supportive + Thiamin
- ↳ Methanol → fomepizole or ethanol
- ↳ Ethyl glycol → fomepizole or ethanol

②

CO poisoning

remove → CPR → O₂ → rest

③

Cyanide poisoning

→ ABC

→ decontamination

→ Antidotes (Amyl nitrate, Sodium Nitrate, Sodium thiosulfate)

④

Opioids overdose → Agonist: methadone
pinpoint pupils ↳ antidote: Naloxone

⑤

CB stimulants: - pupil dilation

Ⓐ Cocaine → No antidote, just supportive

Ⓑ Amphetamine → No antidote, just supportive

⑥

Hallucinogens → cambies

⑦

Insecticides

- garlic odor, dermal irritation, pulmonary edema → symptoms

- Labs → Red Blood cell cholinesterase ↓

- Tx →

• How to asses قوتهم و شدة؟

① stabilization ⇒ Intubation, give O₂

② Decontamination

③ give Atropine

Atropin يمنع عشان هيلك بتعمل overstimulation المبيدات بتعمل

block عشان نهعمل for the receptors

للمستقبلات

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for therapy → IV. 2-4mg

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[M receptors ← Atropin ← حبيب عشان ما نتخرب
N receptors ← pralidoxime ←]

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<p>Take-Home Points</p> <p>"Rule of 150's"</p> <ul style="list-style-type: none"> • >150mg/kg = toxic dose • 7.5g in adults • >150mg/dL at 4 hours <p>NAPQI and NAC: what they do</p> <p>Nomogram for single acute ingestions</p> <ul style="list-style-type: none"> • Very conservative but safe <p>Treatment: indications, timing</p>		

* Benzo Poisoning
Antidote → flumazenil

* Atropin poisoning
Antidote → physostigmine

* Cobalamin toxicity → Antidote Hydrogen cyanide

* Cyanide antidote is → Vitamin B12